A FICTITIOUS NATIVE DISEASE *(ISIGWEBEDHLA)*

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(*Received for publication*, 3 *September*, 1913)

For many years reports have been received from natives and from Europeans resident in Zululand that a serious and often fatal disease, known as Isigwebedhla, appeared regularly in epidemic form about the same time as malaria. The outstanding feature of this complaint was stated to be rapid ulceration of the rectum and anus, described by some as gangrenous rectitis. The disease was said to be characterised by sudden onset and fever. Within three or four days the anus 'ulcerated,' in many cases to such an extent that the closed fist might pass through it with ease, after which the ulceration went on to attack the genitals, and even the mouth and throat. As a rule there was a copious passage of green bile; sometimes, however, mucus tinged with blood was passed, or even grey sloughs. In very severe cases the disease 'attacked the back of the neck.' Blood might appear in the urine, and in rare cases there was haemorrhage from mouth and nose. The condition rarely persisted for any time. In the majority of cases the patient either died, or the disease, even when associated with the most extensive ulceration, rapidly cleared up. This account of the condition was corroborated by practically everyone I interviewed on the subject in Zululand. It must be added, however, that few Europeans had actually seen a case, and their impressions of it were derived from native accounts. Only one post-mortem examination on a fatal case had been made, and Dr. Manning, who performed it, said that he could find no ulceration of the rectum whatever.

On arrival in Zululand I went somewhat further into the local evidence, and summarise the more important particulars below.

(1) Isigwebedhla made its appearance shortly after the arrival of the British in 1823, an event of political significance only.

(2) It occurs annually in epidemic form during and after the rains.

(3) There is a great variation in the severity of the annual epidemic, and in some years it is scarcely noticeable.

(4) This epidemic is always associated with a co-existent epidemic of malaria ('Umkuhlane').

(5) Isigwebedhla occurs only in the bush veldt among the Zulu people, unlike malaria which is endemic on the coast flat among the Tonga and mixed race found there.

(6) It has no relation to the tsetse belts, or to the parts infested with the tick *Ornithodorus moubata* which carries the parasite of relapsing fever.

(7) Dietetic changes, as the use of green mealies, new mabele (millet grain), etc., have no influence on its appearance. The new crop of cereals begins to be used at the time of its greatest incidence.

(8) One attack does not protect against a second, and a person may suffer two, or even more, times from it in the course of a single year.

Accompanied by a posse of native doctors, I watched the progress of nineteen cases, each and all of which were diagnosed by these persons as typical and severe 'Isigwebedhlas.' In eighteen of these I demonstrated either in the peripheral, splenic, or hepatic blood the presence of *Haemamoeba praecox* of malignant tertian malaria. In some instances it was in prodigious numbers. All my cases showed the other cardinal signs of malaria, e.g., pigmented leucocytes and a mononuclear increase, and all recovered rapidly under quinine injections. The clinical types noted were as under:

14 were typical bilious remittents.

2 were of the dangerous algid type with great cardiac weakness and a tendency to collapse.

3 were cerebral in type, 2 showed coma, and the other was characterised by convulsions, delirium and diarrhoea.

True dysentery was not a feature of any of them, although in one case the motions contained blood, but only for a day.

In all, the malarial intoxication was sufficient to markedly lower the tonicity of the sphincter ani, and permit of its easy dilation.

In two cases stated to be exceptionally severe, slight rectal prolapse was present. Both patients were more or less collapsed, and in one the condition was undoubtedly aggravated by a retroverted uterus and two days of irritating diarrhoea.

In all, the rectal mucosa participated in changes similar to those observed in the buccal mucosa in any case of fever. Trifling excoriation was observed in most, and was invariably described by those present as ulceration. No organisms were ever found in the tissues below the 'ulcerations,' and scrapings taken from the parts the natives considered ulcerated showed only the ordinary organisms of the intestinal tract. In the case noted above, in which convulsions were a feature, the diarrhoea was apparently due to a small spirochaete, identical as to staining reactions, and presenting the same relations with the endothelial cells of the gut, as that described by Le Dantec. Certain 'abdominal' features of the case may have been due to this organism, but I do not consider that its importance in this instance as a disease-producing factor compares with the rôle played by the sub-tertian malaria parasite, with which even the peripheral blood was heavily infected. This spirochaete is said to produce a mild diarrhoea, chronic in type, in the South of France, and it is possible it may do the same in Africa. It can be easily overlooked on account of its small size and the difficulty of staining it. Those found by me measure 5 to 10μ by 0·5μ in width. They stain faintly pink by Leishman, and are best shown by weak carbol-fuchsin. They do not take Gram. They are seen as minute coiled threads little longer than a red blood corpuscle. They may occur almost in pure culture, forming in places dense felted networks. A few are seen clinging to the free surfaces of the endothelial cells. They were absent from scrapings taken from the rectal mucosa, and must have had their origin higher up. None of my cases showed *Sp. vincenti*, nor organisms which could be identified with the *Bacillus fusiformis*.

Herpes on the lips, buttocks and perineum was occasionally observed. This is a common concomitant of malaria, and explains the reports of 'ulceration involving the skin.'

In the mouth sores, and those involving the throat, in the only case in which the latter was ulcerated, nothing of interest was seen.

Sp. dentium was once demonstrated, and in two instances I found extensive infection with *Sp. buccalis*.

The absence of eosinophilia in all precluded the likelihood of serious hook worm infection. I searched the dejecta in each case, and failed to find eggs or embryos. Ascarides and tapeworm were common enough. I saw no cases of rectal bilharzia. Routine examination of hepatic blood failed to disclose Leishman-Donovan bodies, and none of these cases showed trypanosomes or *Sp. duttoni*, a matter of some interest when compared to the state of affairs in the low veldt proper, where the existence of Isigwebedhla is denied.

It becomes necessary, however, to explain the apparent discrepancy between native accounts of the disease and the actual facts as determined by my investigation.

The initial treatment of most acute diseases, and specially of specific fevers among the Zulu, consist in taking an enemetic followed by an enema. This is done to remove 'bile.'

Emesis is frequently produced by drinking plain water, but in the majority of instances a host of bulbs, roots and barks pounded up, and used either singly or in combination, are infused in hot water which is then drunk. Most of these substances are harmless, many of them, as the Mfusamvu (*Pittosporum viridiflorum*), act in virtue of their soapy nature, a few are true emetics, and some, as the Macapazane (*Bowiea volubilis*), the juice of which sets up a papular rash on the skin and proves toxic to guinea-pigs in small doses, are irritants.

The rectum is emptied as follows: The patient is placed in the knee-elbow position with the chest resting on the ground. A cow-horn with the point cut off is inserted into the anus and a watery infusion of an extensive series of roots, bulbs, etc., is poured in until the bowel will hold no more, when the enema is evacuated. This process is repeated until the fluid comes away clear, and is continued daily. The enema is often followed by a suppository, or the parts are painted with a decoction of various bulbs. I have been told that in Isigwebedhla a suppository of mud and red chillies is sometimes used, 'with excellent results.'

After one or more days of severe malarial fever the tone of the sphincter ani is so reduced that traction on the buttock of a patient in the knee-elbow position results in an immediate dilatation of the

anus. The degree of patency depends on the success with which one can overcome the resistance of the sphincter, and, other things being equal, varies directly with the gravity of the constitutional effects of the fever. The patency, which in many of my cases was extreme, is to the native mind the 'eating away of the flesh,' i.e., ulceration, and is the one cardinal feature of the 'disease.'

It is customary in these cases to keep the patient sitting up throughout the course of the sickness, to facilitate the easy flow of bile. This is done by relays of friends, and it is not surprising to find the unfortunate patient unable to hold up his head after two or three days. On complaining of pain in the spine or back of the neck, the diagnosis that 'Isigwebedhla' has affected these parts is quickly arrived at.

Malaria is, if anything, more prevalent among the coast people than among the Zulu of the hills. There are places in Hlabisa district where one sees cases of 'Isigwebedhla' in the Zulu kraals among the hills, and hears of dozens in the immediate neighbourhood. Not two miles away on the flat are people who are suffering from malaria in as great a degree, yet deny the existence of 'Isigwebedhla' among themselves, and invariably affirm that it is a condition confined to the Zulu.

I consider that the term 'Isigwebedhla' is an invention of some astute practitioner in the past, and have had some strong hints as to the accuracy of this view of it given me by Tonga 'doctors' whose clientele embraces Zulus. Rightly or wrongly, the Tonga doctor has a greater reputation than his Zulu confrère, and it is not only possible, but likely, that this myth was evolved by the Tonga.

There is no doubt that in judging of the severity of such a case the native is swayed more by his deductions from the constitutional symptoms than by the presence of definite lesions in the rectal mucosa, and he is much influenced by the rapidity with which the constitutional symptoms develop. At the same time he is unable to imagine grave constitutional symptoms existing independently of a gross lesion to account for their production. Given delirium or coma (unless he can explain it by witchcraft) he readily constructs an alarming pathology to account for its existence. A comatose case is 'dead' of Isigwebedhla; he may take a hasty glance at the rectum. If no 'ulceration' is seen, or the patency is not marked,

the former must certainly exist higher up. He would never ascribe symptoms to a blood condition *per se*, even if he could conquer his intuition to ascribe such serious disease to witchcraft, an idea which is strengthened by the absence of gross morbid changes.

Seeing, therefore, that 'Isigwebedhla' is merely an advanced stage of an acute febrile disorder, it is difficult to refrain from admiring the acumen by which these ignorant (sic) practitioners, recognising their utter inability to effect a cure, have evolved this terrible and fatal malady, the successful treatment of which is unexpected at best, and which, if achieved, cannot fail to reflect the highest credit on all concerned in it.